CURRICULUM VITAE

INFORMAZIONI PERSONALI

Nome		**Rossi Antonio**
Anno di nascita		
Qualifica		**PhD**
Amministrazione		
Incarico attuale		**Associate Professor**
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TITOLI DI STUDIO E PROFESSIONALI ED ESPERIENZE LAVORATIVE

Titolo di studio (anno di conseguimento; nome e tipo di istituto di istruzione o formazione)		1985. Master Degree in Biological Sciences at the University of Pavia.
Altri titoli di studio e professionali		1986. Biologist License. 1992. PhD in Biochemistry at the University of Pavia and Genova. 1992-94. Post-doc in Medical Sciences at the University of Pavia.
Esperienze professionali (incarichi ricoperti; data; tipo di azienda o settore; principali mansioni o responsabilità)		1994-2001. Researcher in Biochemistry at the Department of Biochemistry, Medical Faculty of the University of Pavia. 1995-1997. Visiting Researcher at Kinderspital Zurich (Prof. A. Superti-Furga laboratory), University of Zurich, Switzerland. 2001 to date. Associate Professor in Biochemistry at the Department of Molecular Medicine, University of Pavia.
Capacità linguistiche		Italian: mother language English: fluent written and spoken
Attività scientifica (Research interest)		**Molecular basis and therapeutic approaches to osteochondrodysplasias.** Cartilage is a connective tissue that, together with bone, forms the framework supporting the body as a whole. The main functions of cartilage are: i) to provide a framework on which bone deposition may begin through endochondral ossification and ii) to cover joint surfaces, acting as a shock absorber and allowing bones to slide over one another. Chondrodysplasias are a huge family of genetic disorders caused by defects in genes involved in cartilage development and

		homeostasis. These diseases have extensive heterogeneity, therefore they are extremely difficult to diagnose and treat. Our group is focused on disorders caused by defects in the biosynthesis of proteoglycans the main component of the cartilage extracellular matrix, together with collagen. Our current research topics are: 1) biochemical characterization of new skeletal dysplasias caused by defects in proteoglycan biosynthesis; 2) deep phenotyping of validated animal models for Diastrophic dysplasia and Desbuquois dysplasia type 1; 3) development of potential therapeutic approaches to chondrodysplasias using animal models; 4) characterization of the osteoarthritic phenotype in animal models of skeletal disorders and comparison to common osteoarthritis. Different methodological approaches are used including generation and characterization of murine models, cell cultures, expression studies at the RNA and protein level, histology, confocal microscopy, X-rays and microCT. Our research will deliver new information on the molecular basis of cartilage disorders immediately leading to an increased chance to translate this knowledge into therapies for patients. What we build and what we learn from rare diseases is extensible to common diseases, not only for the immediate goal of better diagnoses, but also for the long-term challenge of identifying drug targets for common diseases (i.e. osteoarthritis).

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